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TRANSMISSION DYNAMICS OF HIV/AIDS WITH SCREENING AND NON-LINEAR INCIDENCE

Kung'aro, M.[1], Massawe, E. S.[1] and Makinde, O.D.[2]

[1]Mathematics Department, University of Dar es Salaam,
P. O. Box 35062, Dar es Salaam, Tanzania
[2] Institute for Advance Research in Mathematical Modelling and Computations,
Cape-Peninsula University of Technology, P. O. Box 1906, Bellville 7535, South Africa

ABSTRACT: *This paper examines the transmission dynamics of HIV/AIDS with screening using non-linear incidence. A nonlinear mathematical model for the problem is proposed and analysed qualitatively using the stability theory of the differential equations. The results show that the disease free equilibrium is locally stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. Globally, the disease free equilibrium is not stable due existence of forward bifurcation at threshold parameter equal to unity. However numerical results suggest that screening of unaware infectives has the effect of reducing the transmission dynamics of HIV/AIDS. Also, the effect of non-linear incidence parameters showed that transmission dynamics of HIV/AIDS will be lowered when infectives after becoming aware of their infection, do not take part in sexual interaction or use preventive measures to prevent the spreading of the infection. Numerical simulation of the model is implemented to investigate the sensitivity of certain key parameters on the transmission dynamics of HIV/AIDS with screening using non-linear incidence.*

Keywords*: HIV/AIDS, Screening, Non-linear incidence, Reproduction number, Stability*

INTRODUCTION

HIV/AIDS has become a global problem. Its infection which emerged in 1981 has become a famous sexual transmitted disease throughout the world. It has started getting attention as it has become a death sentence and fear to a lot of people mainly because there is no cure available till to date (Naresh, *et al,* 2008).

HIV belongs to a class of viruses known as retroviruses, which contain ribonucleic acid (RNA) as their genetic material. After infecting a cell, HIV uses an enzyme called reverse transcriptase to convert its RNA into deoxyribonucleic acid (DNA) and then proceeds to replicate it using the cells machinery (Anderson and May R., 1991).

Early detection of HIV through voluntary screening is important for intervention and for reducing HIV transmission. Early identification can provide the opportunity for timely treatment of infected individuals, thus reducing morbidity and mortality. Additionally, the decrease in risky behaviour resulting from HIV counselling and the reduction in infection due to the use of antiretroviral therapy (ART) can translate into a significant benefit from reduced HIV transmission (Tole *et al*, 2009). However, increasing the number of people who know their HIV status especially among most at risk populations through HIV testing and counselling is key to expanding access to HIV prevention, treatment and care. The fundamental principle of HIV testing is that it must be accompanied by basic pre-test information to enable the client make an informed and voluntary decision to be tested.

The essential assumption in most classical compartmental epidemic models is that the individuals are homogeneously mixed and each individual has the same chance of getting infected when a small number of infectives are introduced to the susceptible populations (Yuan and Wang, 2009). The rate of new infections, known as the incidence rate, thus takes the bilinear form (mass action). In reality, populations may not be homogeneously mixed and thus it is more realistic to take heterogeneities in population mixing into consideration in modelling the spread of infectious diseases.

It has been suggested by several authors that the disease transmission process may have nonlinear incidence rate. This allows one to include behavioural changes and prevent unbounded contact rates (Liu *et al*, 1987; Moghadas and Gumel, 2002). A particular example of such an incidence rate is given by $\alpha I^s \Big/ \left(1+\beta I^k\right)$, with $s,k,\alpha,\beta > 0$ (Kyrychko and Blyuss, 2005).

Tripathi *et al*. (2007) established and analyzed a mathematical model on the effect of screening of unaware infectives on the spread of HIV infection. However the integration of screening using non-linear incidence rates was not incorporated. In this paper, it is therefore intended to establish and analyze a model which will incorporate the aspect of non-linear incidence in assessing transmission dynamics of HIV/AIDS with screening. We thus study and analyze a deterministic model of Transmission Dynamics of HIV/AIDS with screening using non-linear incidence rates. The model assumes that susceptibles become infected via sexual contacts with both types of infectives and individuals will die due to disease after reaching the full blown AIDS stage.

MODEL FORMULATION

In modelling the disease dynamics, the population is subdivided into four population compartments depending on the HIV status of the individuals: The susceptibles or HIV negatives $S(t)$, HIV positives or infectives who do not know whether they are infected $I_1(t)$, HIV positives or infectives who know that they are infected $I_2(t)$ and those with full blown AIDS $A(t)$ (Tripathi *et a*l, 2007).

In formulating the model, the following assumptions are taken into consideration:

(i) The mode of transmission is assumed to be via heterosexual contacts as this represents the single major primary mode of HIV infection globally,

(ii) Susceptible individuals are considered to be heterogeneously mixed, and it is assumed that unaware and aware infectives will move to full blown AIDS at the rate δ,

(iii) Unaware infectives can be transferred to aware infective class after screening by the rate θ.

(iv) Unaware infectives and aware infectives can infect susceptibles at different rates β_1 and β_2 respectively,

(v) Individuals will die due to disease after reaching the full blown AIDS stage by the rate α,

(vi) The population under consideration comprise persons with at least 18 years of age,

(vii) All parameters and variables of the model are considered to be positive.

Taking into account of the above consideration, we then have the following transfer diagram of the model:

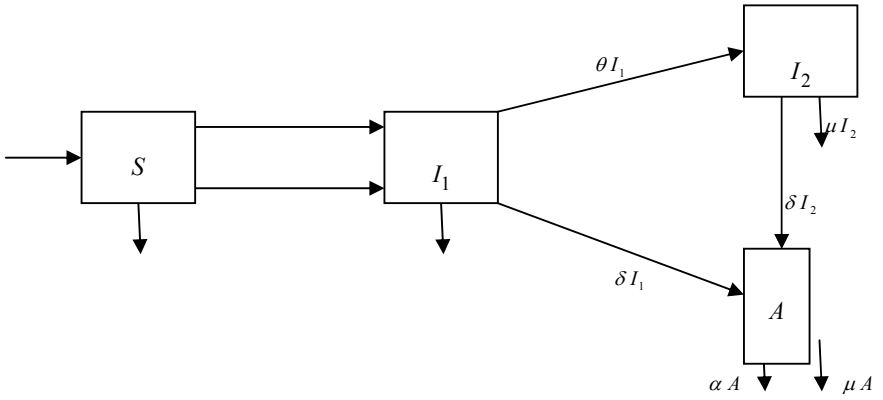

Figure 1: A compartmental model for transmission dynamics of HIV/AIDS

The model is thus governed by the following system of non linear ordinary differential equations:

$$\frac{dS}{dt} = \lambda N - \left[\frac{\beta_1 I_1}{N + a_1 I_1} + \frac{\beta_2 I_2}{N + a_2 I_2}\right] S - \mu S$$

$$\frac{dI_1}{dt} = \left[\frac{\beta_1 I_1}{N + a_1 I_1} + \frac{\beta_2 I_2}{N + a_2 I_2}\right] S - (\theta + \delta + \mu) I_1 \quad (1)$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu) I_2$$

$$\frac{dA}{dt} = \delta I_1 + \delta I_2 - (\alpha + \mu) A$$

with nonnegative initial conditions

$$S(0) > 0,\ I_1(0) \geq 0,\ I_2(0) \geq 0,\ A(0) > 0$$

where

λ is the recruitment rate,

α is the disease (AIDS) related death rate,

μ is the background mortality rate unrelated to HIV/AIDS,

θ is the transfer rate from the asymptomatic to the symptomatic compartment,

δ is the AIDS progression rate,

$\beta_i\ (i = 1, 2)$ are the per capital contact rates for susceptible with unaware infectives and aware infectives respectively,

$a_i\ (i = 1, 2)$ are non-linear incidence parameters with respect to unaware and aware infectives respectively.

The total population at any time t is then given by

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t).$$

It is reasonable to assume that $\beta_2 < \beta_1$ because on becoming aware of the infection, one may choose to use preventive measures and change behaviour.

Since the variable A of system (1) does not appear in the first three equations, in the subsequent analysis we can analyze qualitatively the following subsystem (Cai *et al*, 2009):

$$\frac{dS}{dt} = \lambda N - \left[\frac{\beta_1 I_1}{N + a_1 I_1} + \frac{\beta_2 I_2}{N + a_2 I_2}\right] S - \mu S$$

$$\frac{dI_1}{dt} = \left[\frac{\beta_1 I_1}{N + a_1 I_1} + \frac{\beta_2 I_2}{N + a_2 I_2}\right] S - (\theta + \delta + \mu) I_1 \quad (2)$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu) I_2.$$

In terms of the total population N, the subsystem model becomes

$$\frac{dN_1}{dt} = \lambda N - \mu N_1 - \mu(I_1 + I_2)$$

$$\frac{dI_1}{dt} = \left[\frac{\beta_1 I_1}{N + a_1 I_1} + \frac{\beta_2 I_2}{N + a_2 I_2}\right] S - (\theta + \delta + \mu) I_1 \quad (3)$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu) I_2$$

where

$$N_1(t) = S(t) + I_1(t) + I_2(t)$$

The system (3) is well posed for $N_1(0) > 0$, since solutions remain the non-negative initial conditions.

Model analysis

The nonlinear system in Equation (3) will be qualitatively analyzed so as to find the conditions for existence of stability disease free equilibrium points (Gomes *at el*, 2004). Analysis of the model allows us to determine the impact of screening and non-linear incidence. Threshold condition(s) which govern elimination or persistence of HIV/AIDS transmission will be determined and studied. Also on finding the reproductive number R_0 one can determine if the disease become endemic in a population or not.

Positivity of solutions

It is necessary to prove that all solutions of system (3) with positive initial data will remain positive for all times $t > 0$. This will be established by the following theorem.

Theorem 1

Let $S(0) > 0$, $I_1(0) \geq 0$, $I_2(0) \geq 0$, . Then solutions $S(t)$, $I_1(t)$ and $I_2(t)$ of system (3) are positive $\forall t \geq 0$.

Proof

To prove theorem 1, we use all equations of the model (3). From the system (3), we obtain the inequality expression

$$\frac{dI_1}{dt} \geq -(\theta + \delta + \mu) I_1$$

which gives

$$I_1(t) \geq C \exp\{-(\theta + \delta + \mu)t\} > 0.$$

As $t \to \infty$ we obtain $0 \leq I_1(t) \leq 1$. Hence all feasible solution of system (3) enter region $\Gamma = \{(S, I_1, I_2,)\}$. Similar proofs can be established for the positivity of the other solution.

Disease-free equilibrium point (DFE) and its stability

The disease free equilibrium of the model (3) is obtained by setting

$$\frac{dS}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = 0.$$

(4)

At disease-free equilibrium, we have

$$I_1 = I_2 = 0$$

so that model (3) becomes

$$\lambda N - \mu S = 0$$

(5)

Therefore, the disease-free equilibrium (DFE) denoted by E_0 of the model (3) is given by

$$E_0 = (S,\ 0,\ 0) = \left(\frac{\lambda N}{\mu},\ 0,\ 0\right),\ \mu > 0$$

(6)

Local stability of DFE

The disease free equilibrium of the model (3) was given by

$$E_0 = (S,\ 0,\ 0) = \left(\frac{\lambda N}{\mu},\ 0,\ 0\right)$$

(7)

In order to assess the local stability of the E_0 established by next generation method on the system (3), computation of basic reproduction number is essential. The basic reproduction number R_0 is defined as the effective number of secondary infections caused by typical infected individual during his entire period of infectiousness (Diekman *et al*, 1990). This definition is given for the models that represent spread of the infection in a population. It is obtained by taking the dominant eigenvalue (spectral radius) of

$$\left[\frac{\delta F_i(E_0)}{\delta x_j}\right] \cdot \left[\frac{\delta V_i(E_0)}{\delta x_j}\right]^{-1}$$

(8)

where

F_i is the rate of appearance of new infection in compartment i,

V_i^+ is the transfer of individuals into compartment i,

V_i^- is the transfer of individuals out of compartment i by all other means,

E_0 is the disease-free equilibrium.

Consequently

$$\begin{pmatrix} f_1 \\ f_2 \end{pmatrix} = \begin{pmatrix} \frac{\beta_1 I_1 S}{N + a_1 I_1} + \frac{\beta_2 I_2 S}{N + a_2 I_2} \\ 0 \end{pmatrix}$$

By linearization approach, the associated matrix at disease-free equilibrium is given by

$$\mathbf{F}=\begin{pmatrix}\frac{\delta f_1}{\delta I_1}(E_0) & \frac{\delta f_1}{\delta I_2}(E_0)\\ \frac{\delta f_2}{\delta I_1}(E_0) & \frac{\delta f_2}{\delta I_2}(E_0)\end{pmatrix} \tag{9}$$

which gives

$$\mathbf{F}=\begin{pmatrix}\frac{\beta_1\lambda}{\mu} & \frac{\beta_2\lambda}{\mu}\\ 0 & 0\end{pmatrix}$$

and

$$\begin{pmatrix}v_1\\ v_2\end{pmatrix}=\begin{pmatrix}(\theta+\delta+\mu)I_1\\ -\theta I_1+(\delta+\mu)I_2\end{pmatrix}$$

Again by linearization we get

$$\mathbf{V}=\begin{pmatrix}\frac{\delta v_1}{\delta I_1}(E_0) & \frac{\delta v_1}{\delta I_2}(E_0)\\ \frac{\delta v_2}{\delta I_1}(E_0) & \frac{\delta v_2}{\delta I_2}(E_0)\end{pmatrix}$$

yielding

$$\mathbf{V}=\begin{pmatrix}\theta+\delta+\mu & 0\\ -\theta & \delta+\mu\end{pmatrix}$$

with

$$\mathbf{V}^{-1}=\begin{pmatrix}\frac{1}{\theta+\delta+\mu} & 0\\ \frac{\theta}{(\theta+\delta+\mu)(\delta+\mu)} & \frac{1}{\delta+\mu}\end{pmatrix}.$$

Therefore

$$\mathbf{FV}^{-1}=\begin{pmatrix}\frac{\beta_1\lambda}{\mu(\theta+\delta+\mu)}+\frac{\beta_2\lambda\theta}{\mu(\theta+\delta+\mu)(\delta+\mu)} & \frac{\beta_2\lambda}{\mu(\delta+\mu)}\\ 0 & 0\end{pmatrix}$$

The eigenvalues of $\mathbf{FV}^{-1}$ are

$$\left(0,\ \frac{\beta_1\lambda}{\mu(\theta+\delta+\mu)}+\frac{\beta_2\lambda\theta}{\mu(\theta+\delta+\mu)(\delta+\mu)}\right)$$

The basic reproduction number for the model (3) with non-linear incidence denoted by R_0 is

$$R_0=\frac{\beta_1\lambda(\delta+\mu)+\beta_2\lambda\theta}{\mu(\theta+\delta+\mu)(\delta+\mu)} \tag{10}$$

Thus the disease free equilibrium of the transmission dynamics of HIV/AIDS model (3) with screening and non-linear incidence is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$. This can be realized when one tries to assess the contribution of I_1 and I_2 in terms of β_1 and β_2 respectively from equation (10) above. Let

$$R_{0a}=\frac{\beta_1\lambda}{\mu(\theta+\delta+\mu)} \text{ and}$$
$$R_{0b}=\frac{\beta_2\lambda\theta}{\mu(\theta+\delta+\mu)(\delta+\mu)} \tag{11}$$

where

$$R_0=R_{0a}+R_{0b}. \tag{12}$$

It is clear from equations (11) that $R_{0a}>R_{0b}$ which implies that for large infective population, the unaware infectives I_1 have a significant contribution on the transmission of the infection and keep the disease endemic in the population via β_1 compared to aware infectives I_2 via β_2.

Endemic equilibrium

To find endemic equilibrium, denoted by $E_1(I_1^*,I_2^*,S_1^*)$, we set the right hand side of each equation of the system (3) equal to zero and express the other dependent variables in terms of I_1^* at equilibrium point and to obtain

$$I_2^*=\frac{\theta I_1^*}{(\delta+\mu)},$$

Substituting I_2^* and S^* in the second equation of (3) at steady state, we obtain after some calculations that I_1^* must satisfy the following equation

$$S^* = \frac{\left[N(\delta+\mu)+a_2\theta I_1^*\right]\left[N+a_1 I_1^*\right]\lambda N}{I_1^{*2}\left(\mu a_1 a_2\theta+\beta_2 a_1\theta+\beta_1 a_2\theta\right)+I_1^*\left(\mu N\theta a_2+\mu N\delta a_1+\mu^2 N a_1+N\delta\beta_1+N\beta_1\mu\right)+N^2\left(\mu\delta+\mu^2\right)} \tag{12}$$

$$I_1^* f\left(I_1^*\right) = I_1^*\left(AI_1^{*2}+BI_1^*+C\right)=0 \tag{13}$$

where

$$A = \beta_1 a_2\theta^2+\mu a_1 a_2\theta^2+\beta_2 a_1\theta^2+\mu^2 a_1 a_2\theta+\delta\mu a_1 a_2\theta+\delta\beta_1 a_2\theta$$
$$+\delta\beta_2 a_1\theta+\mu\beta_1 a_2\theta+\mu\beta_2 a_1\theta$$

$$B = \beta_2 N\theta^2+\mu N a_2\theta^2+\beta_1 N\delta^2+\mu^3 a_1 N+\beta_1 N\mu^2+\delta^2 a_1\mu N$$
$$+\mu^2 N a_2\theta+\mu a_1 N\delta\theta+\mu^2 N a_1\theta+\beta_1\theta N\delta+\beta_1 N\mu\theta+\delta\mu N a_2\theta$$
$$+2\delta a_1 N\mu^2+2\delta\beta_1 N\mu+\delta\beta_2\theta N+\mu\beta_2\theta N-\theta\beta_1\lambda N a_2-\theta\beta_2\lambda N a_1$$

$$C = \mu N^2\delta^2+2\delta N^2\mu^2+\theta N^2\mu^2+N^2\mu^3+\theta\mu N^2\delta$$
$$-\beta_1\lambda\delta N^2-\beta_1\lambda\mu N^2-\beta_2\theta\lambda N^2$$

$$f_1\left(I_1^*\right)=\frac{\beta_1 I_1^*}{N+a_1 I_1^*}$$

From equation (13) it can be seen that the root for $I_1^*=0$ corresponds to the DFE. The relationship $f\left(I_1^*\right)=0$ corresponds to the existence of multiple equilibria. The model also exhibits a forward bifurcation for some estimated parameters as seen in figure 2 below:

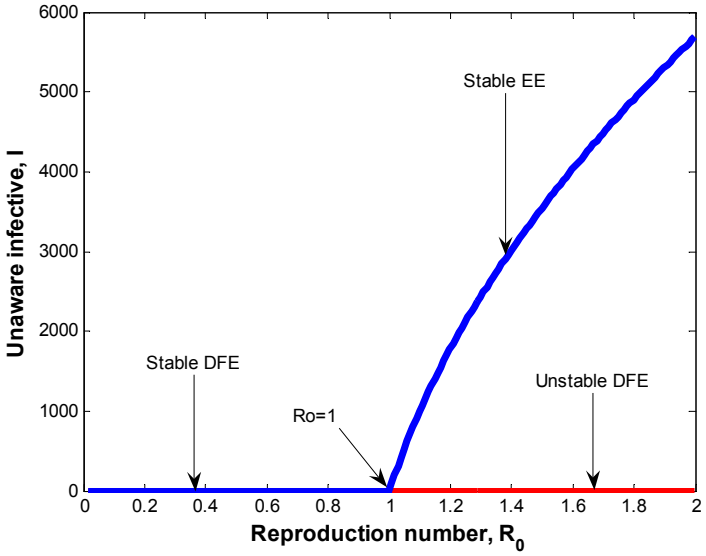

Figure 2 Forward bifurcation in the (R_0, I) plane

From the bifurcation figure, it can be seen that, the EE point being locally asymptotically stable, the disease can invade the population and transmission dynamics can persist if no purposive interventional measures are subjected to the population for the purpose of reducing the disease or if possible to eradicate it. Hence control of the epidemic depends on the enhancement of the behavioural changes among the subgroups of populations where the spreading of the disease occurs and optimum use of the available therapy for those infected.

Global stability analysis

The global stability analysis of the epidemic models is generally difficult to carry out. Consequently, the literature on global analysis of dynamical systems is very little. Busenberg and van den Driessche (1990) proposed an elegant technique for proving the non-existence of certain type of solutions such as periodic orbits, homoclinic orbits and polygons associated with SIR models. In this regard, using the Busenberg and van den Driessche technique it can be shown that the global stability analysis of model (3) is given as follows. We set

$$\Gamma_1 = \left\{ (S, I_1, I_2) \in \Gamma : S + \frac{(\mu+\delta)}{\mu} I_1 + \frac{(\mu+\delta)}{\mu} I_2 > \frac{\lambda N}{\mu} \right\}$$

$$\Gamma^* = \left\{ (S, I_1, I_2) \in \Gamma : S + \frac{(\mu+\delta)}{\mu} I_1 + \frac{(\mu+\delta)}{\mu} I_2 = \frac{\lambda N}{\mu} \right\}$$

$$\Gamma_2 = \left\{ (S, I_1, I_2) \in \Gamma : S + \frac{(\mu+\delta)}{\mu} I_1 + \frac{(\mu+\delta)}{\mu} I_2 < \frac{\lambda N}{\mu} \right\}$$

Thus $\Gamma_1, \Gamma^*, \Gamma_2$ are pair wise disjoint subsets of Γ, and $\Gamma = \Gamma_1 \cup \Gamma^* \cup \Gamma_2$.

Let $N_1 = S + I_1 + I_2,\ (S, I_1, I_2) \in \Gamma$. From system (3), the equation for the total population N_1 satisfies

$$\frac{dN_1}{dt} = \lambda N - \mu N_1 - \delta (I_1 + I_2). \tag{14}$$

Consequently, in $\Gamma_1, \Gamma^*, \Gamma_2$, we have $\frac{dN_1}{dt} > 0,\ \frac{dN_1}{dt} = 0,\ \frac{dN_1}{dt} < 0$, respectively. It then follows that Γ^* is a positively invariant set in Γ.

Numerical simulations

In order to illustrate some of the analytical results of the study, numerous numerical simulations of the model (3) are carried out using a set of reasonable parameter values given in table 1 below and the following estimated initial conditions $S = 500,\ I_1 = 250,\ I_2 = 100$

However these parameters may (or may not) be biologically feasible.

Figure 4.1 below shows variation of the S, I_1 and I_2 with time when $R_0 = 0.7058$,

Table 1: Parameter values used in numerical simulations

Parameter symbol	Parameter value $(yr)^{-1}$	Source
λ	0.0100	Estimated
μ	0.0200	Tripathi *et al* (2007)
θ	0.6000	Issa *et al* (2010)
δ	0.0500	Nyabadza *et al* (2010)
α	1.0000	Naresh *et al* (2009)
a_1	0.0900	Estimated
a_2	0.0100	Estimated
β_1	0.8600	Estimated
β_2	0.1500	Tripathi *et al* (2007)

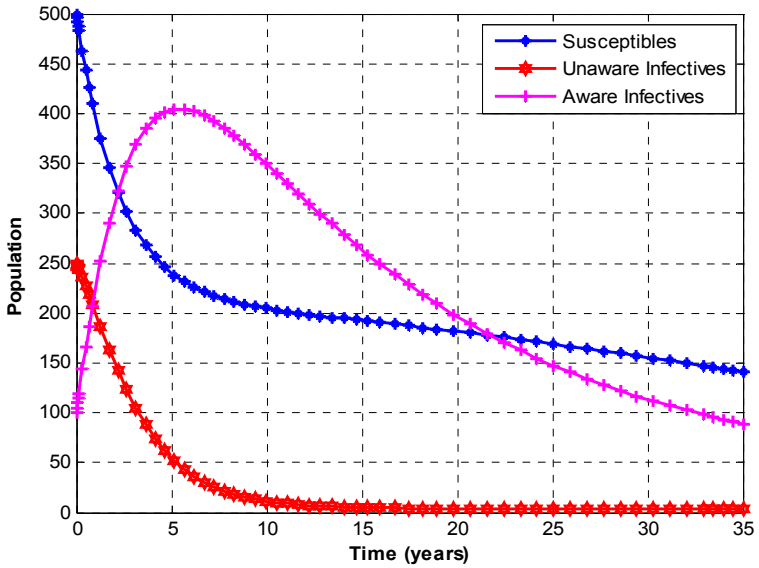

Figure 4.1: General variation of population in different classes

In Figure 4.1 it is seen that the number of susceptibles and unaware individuals decrease with time. Initially aware infective class increase with time and then reaches its equilibrium position. This is due to an increase in screening rate in which unaware individuals are moved to this class by the rate θ. This explains why unaware infectives decrease rapidly to zero, susceptibles decrease to a certain level but do not diminish to zero. This means that we can control the epidemic through promoting behavioural change and taking necessary precautions while having sexual interaction.

As it can be seen in Figure 4.2, when screening rate becomes zero, the infectives who do not know that they are infected, continue maintaining sexual relationship in the community leading to persistance of the disease as far as $R_0 > 1$. But when the rate of screening increases, there is a possibility of the disease to cease because individuals may use prenventive measures after knowing their HIV status.

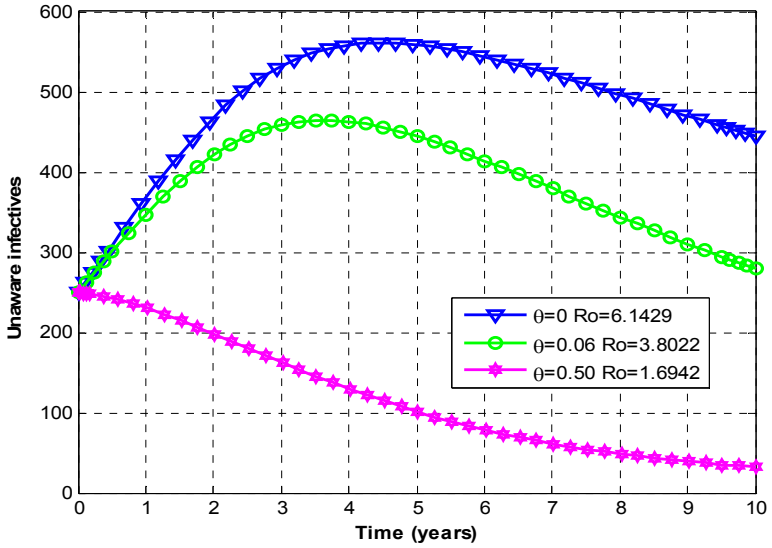

Figure 4.2 : Variation of unaware infectives with time for different values of θ

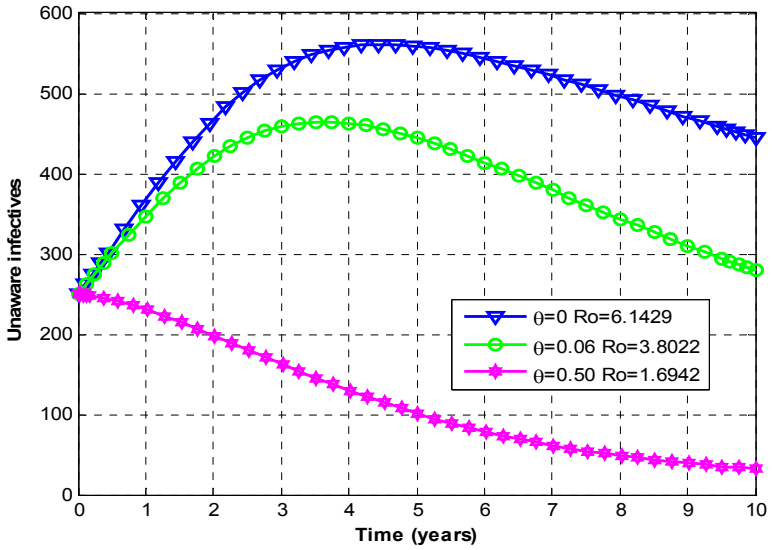

Figure 4.3 : Variation of aware infectives with time for different values of θ.

Figure 4.3 reveals that if screening rate θ is increased, aware (symptomatic) individuals also increase. Symptomatic infectives after knowing their HIV status may change their behaviour and continue surviving and thus reduce the AIDS population. It is therefore suggested that, to minimize the spread of the disease, the population under consideration should be encouraged to attend medical screening for the purpose of changing their behaviour and use preventive measures.

Figures 4.4 (a) and 4.4(b) above show the role of contact rate β_2 of aware HIV infectives and susceptible respectively. It can be seen that as aware HIV infectives continue maintaining sexual interaction without exposing themselves, the susceptible population decrease rapidly leading to an increase in aware HIV infectives thus increasing transmission dynamics and hence disease persists in the population as the number of AIDS individuals increase.

The effect of non-linear incidence parameter $a_{i=1,2}$, are observed in the following figures using the following estimated initial conditions $S = 1500,\ I_1 = 700$ and $I_2 = 300$.

Figure 4.5 assess the impact of non-linear incidence parameters against susceptibles. As it can be seen from the figure, the susceptibles decrease very slowly with time for a very high non-linear incidence rate. This means that if the infected individuals manage to change their behaviour and stop spreading the disease, the epidemic can go to extinction as long as very few susceptibles are infected.

Figure 4.6 shows that infectives who are not aware of their status decrease rapidly whenever saturation effects are achieved, that is conditions under which the disease can persists in the population are well known to them. Intervention strategies are then used to infected individuals and behavioural changes are well achieved within the society.

The rate of new infection plays a very important role in the investigation of disease dynamics. As seen in figure 4.7, if aware infected individuals do not interact with others, that is, non-linear incidence rate is zero, the number of infected individuals increase rapidly with time hence increasing transmission dynamics. However, the speed of increasing changes if infected individuals interact with others and positive response to intervention programs are effective.

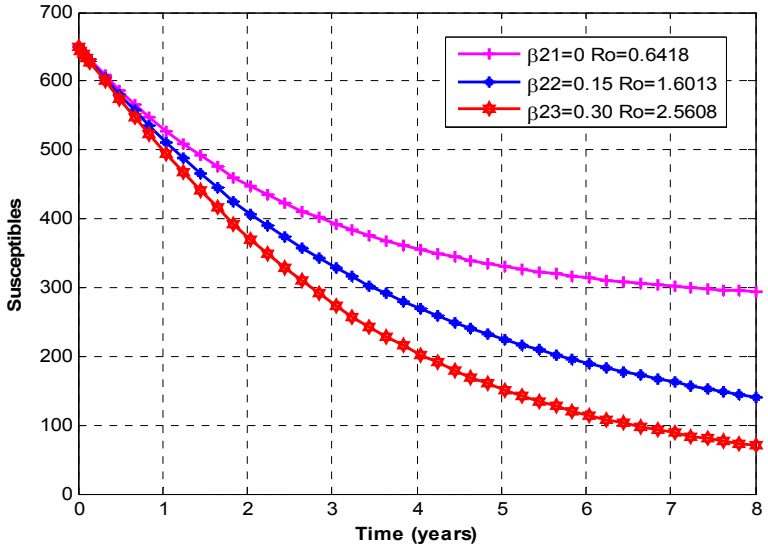

Figure 4.4(a) Variation of sucseptibles for different values of β_i

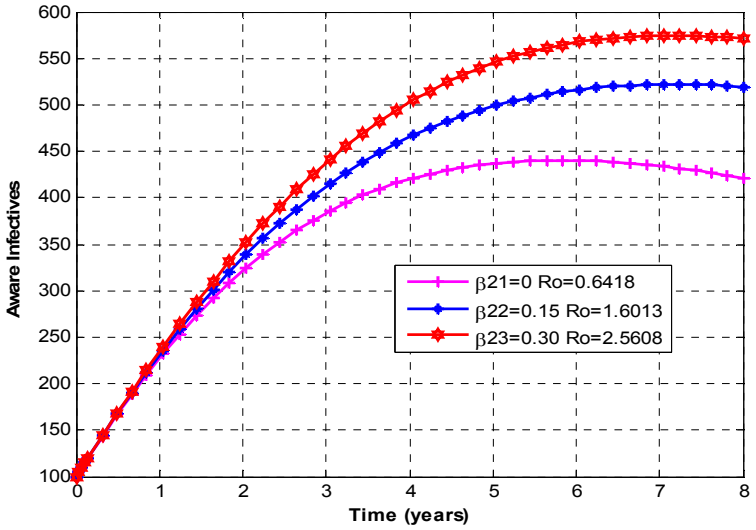

Figure 4.4(b): Variation of aware infectives for different values of β_i

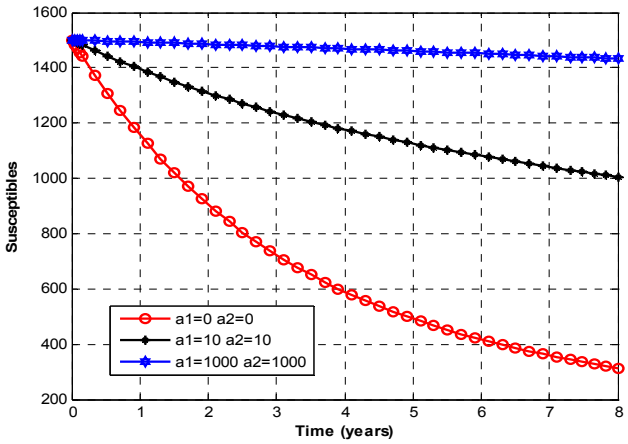

Figure 4.5: Variation of susceptible individuals for different values of $a_{i=1,2}$

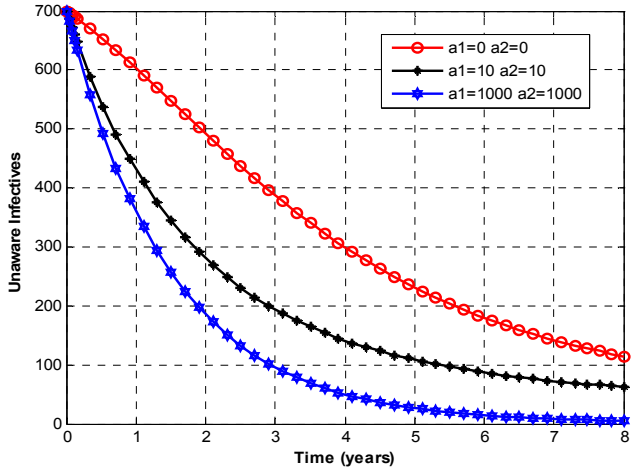

Figure 4.6: Variation of unaware infectives for different values of $a_{i=1,2}$

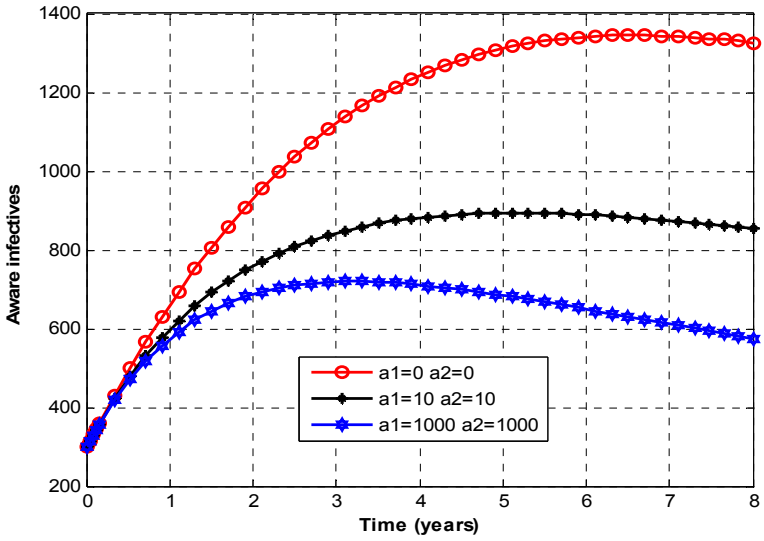

Figure 4.7: Variation of aware infectives for different values of $a_{i=1,2}$

Thus, to maintain the spread of the epidemic at control, the detected infective individuals should be provided with thorough education with respect to their behavioural changes so as to either abstain from sexual interaction or use preventive measures to stop spreading the infection.

DISCUSSION AND CONCLUSION

In this paper, a non-linear mathematical model has been established to study the transmission dynamics of HIV/AIDS with screening and non-linear incidence. The main objective of the study was to assess the transmission dynamics of HIV/AIDS with screening using non-linear incidence. In the study it was assumed that there is no vertical transmission of the disease and mode of transmission is assumed to be via heterosexual contacts. Susceptibles are considered to be heterogeneously mixed and disease related death rate is assumed to occur to individuals after reaching the full-blown AIDS stage. Both qualitative and numerical analyses of the model were done. Qualitative analysis of the model involved computation of the basic reproduction number. The model showed that the disease free equilibrium is locally stable at threshold parameter less than unity and unstable at threshold parameter greater than unity, but globally the disease free equilibrium is not stable due existence of forward bifurcation at threshold parameter equal to unity. Also the model analysis showed the existence of unique endemic equilibrium, that is, locally stable under certain conditions when the threshold parameter exceeds unity due to existence of forward bifurcation at threshold parameter equal to unity. The endemic equilibrium is found to be globally stable under certain conditions.

A numerical study of the model was performed to see the effects of certain key parameters on the spread of the disease. The analysis shows that the screening of unaware HIV infectives and treatment of screened HIV infectives have the effect of reducing the transmission of the disease. It is observed that when the screened infectives and treated infectives do not participate in the transmission of the infection, the AIDS population is significantly reduced in comparison to the case where there is no screening and treatment. In the absence of screening, the endemicity of the infection increases results in the increase of AIDS population.

Based on the results of the study, it is concluded that the most effective approach that can be used to possibly reduce transmission dynamics of the disease and lower the incidence rate is emphasis on information campaign in order to reduce HIV prevalence. Furthermore people should be educated and be aware of preventive measures for the the spread of the disease to be under control. Thus, education campaign must reach the community at all social levels, especially in lower classes and to the high risk groups so as to increase the awareness about the disease and protection measures so as to enhance the control of the disease. The HIV/AIDS eradication remains a challenge to all parts of the world particularly in most developing countries. Hence, there is a need to strengthen the control strategies at hand as well as putting more emphasis on the behavioural changes among individuals.

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